

ISCHEMIC MUSCULAR ATROPHY, CONTRACTURES AND PARALYSIS.*

BY ALEXANDER HUGH FERGUSON, M.D., C.M.,

OF CHICAGO, ILL.,

Professor of Clinical Surgery in the University of Illinois.

ISCHEMIC atrophy and paralysis, being the changes which occur in muscles from which the blood-supply is more or less cut off, may be due to the following causes:

I. *Arterial*—depending on two causes: (a) Interruption must be nearly complete; (b) interruption must be of more than two of three hours' duration, otherwise it is easily tolerated—sometimes incomplete interruption for several days.

II. *An interrupted venous return*.—Causes: 1. Embolus—cardiac; 2. Thrombus—syphilitic endarteritis, or following acute infectious diseases; 3. Raynaud's disease—changes in vessels due to defective nerve innervation; 4. Direct injury to a vessel; 5. Cold; 6. Most common of all—tight splinting, to which I shall mostly refer.

Symptoms.—Paresthesia—numbness and tingling in parts of limb affected, and perhaps combined with alternating sensations. Severe and often paroxysmal pain in muscles followed by cramps and spasmodic jerkings. Limb often pale, but sometimes cyanosed and cold. Electric irritability of muscle lost after ischemic condition has lasted five hours, and the muscles are quite flaccid and powerless, as seen in operations after prolonged use of Esmarch's bandage. After seven hours, muscular rigidity and painful contractures begin. These increase in severity, but disappear in two or three days, leaving muscle again flaccid. Edematous condition from more or less established collateral circulation. Muscles now tender on press-

* Read before the Chicago Surgical Society, December 1, 1905.

ure. Swelling gradually disappears; muscular atrophy becomes manifest, muscles being hard and firm.

Contractures follow—hand flexed at wrist, and fingers at phalangeal joint. Final contractures due to atrophy of muscles and overgrowth of fibrous connective tissue. Atrophic changes in the skin often present.

Course.—This depends on the duration of the primary ischemic condition. When cramps and rigidity have set in, probably no immediate recovery of the muscle is to be looked for, but short of this the muscles may readily recover, if the blood supply is restored. In cases where cramps have appeared, it is probable that many of the muscular fibres die, and have to be replaced by regeneration—a long and slow process, or by repair. When ischemia is of greater duration, and the muscles have become flaccid, it is not probable that any great amount of restitution can occur. However, certain fibres may have escaped, and from these new fibres may be formed, but the amount of recovery will be small. In such cases the muscles rapidly atrophy, becoming tough, hard and contracted.

Pathological Anatomy.—A. If blood supply is completely interrupted. B. If blood supply is partly interrupted.

A.—1. Muscle is gray, dirty yellow color, dull in appearance, and friable.

2. Under microscope—transverse and longitudinal structures more obvious.

3. Almost complete absence of muscular nuclei.

4. Usually no fatty degeneration.

B.—1. Muscle firm and tough; more or less normal in appearance.

2. Under microscope—great increase of fibrous connective tissue between bundles of muscular tissue and between individual muscle, fibres and cells.

3. Fibres have lost their polygonal form; are smaller and rounder, but maintain their striæ even under extreme atrophy.

II. *Interrupted Venous Return.*—1. Acute symptoms are

those of thrombus, viz., swelling, cyanosis, œdema; limb feels fuller, harder, and is tender on pressure.

2. Acute symptoms may pass off, but limb is left in condition of passive congestion, making limb larger—pseudo-hypertrophy.

Pathological Anatomy.—1. Early stage—increase of intermuscular fibrous connective tissue.

2. Later stage—connective tissue more abundant; some atrophy of muscles, fibres and fatty degeneration.¹

Pressure Lesions of Nerves.—1. The resultant symptoms of severe cases of this are similar to nerve section and are paralytic in nature, pain and hyperæsthesia being usually absent.

2. Paralysis usually more marked than anæsthesia.

3. Trophic changes are usually rare.²

Having now made a brief, and, I trust, concise review of the most prominent points relating to ischemia, I desire to add a short account of two recent cases of this nature, on account of their aptitude to the subject, and the surprisingly favorable results attending my operative treatment of them.

CASE I.—On the 4th day of May, 1902, a school-boy, aged nine years, and weighing 65 pounds, entered Chicago Hospital, with the following trouble:

(a) Deformity of hand. The forearm, wrist and hand were crippled, and abnormal in size, shape and color. The whole extremity below the elbow was apparently much smaller than normal, and the fingers, which looked blue and felt cold, were flexed into the palm.

(b) Fixation. The fingers were not merely flexed into the palm, as already stated, but also there fixed. No effort on his part, nor considerable force by me, could effect but the slightest

¹ Chiefly indebted for this data to F. E. Batten, M.D., F.R.C.P., London.

² Chiefly from an article by W. Thorburn, M.D., and R. T. Williamson, Manchester, England.

increased flexion or extension. Voluntary motion at the wrist, or supination, or pronation, was extremely limited. (Fig. 1.)

(c) Atrophy. The bellies of the muscles appeared to be entirely absent, except near the elbow. The skin hugged the bones; the deeper soft parts were firm, hard and immovable. The circumference of the forearm was less than that of its fellow by about one-third.

(d) Paralysis. The hand was paralyzed as to motion, although the muscles responded to irritation and electricity near the elbow at the points where they were applied. When electricity was applied to the extensor muscles of the hand, no extension ensued of either wrist or fingers. Sensation of the fingers and hand was rather increased. Sensations of heat and cold were normal.

(e) Loss of function. The hand was useless for ordinary purposes.

Although an antecoperative photograph was not successfully obtained, yet I endeavored to make good this interesting deficiency by having a postoperative picture taken of the arm placed in its original malposition. This is shown in Fig. 1.

History.—Four years previously the patient had both bones of right forearm broken at the middle, which, when set, was put up with fingers flexed and bandages tight. When the splints were removed, seven weeks afterwards, and the condition of the limb discovered, he was brought to an eminent Chicago surgeon, who stretched the fingers, giving some improvement, but no cure.

Operation.—An Esmarch bandage was applied in proper manner for the case. A long curved incision was made on flexor surface of forearm. All the muscles and tendons were found matted together by fibrous tissue. These were separated from each other, and the ulnar and median nerves were disengaged and stretched. Muscle-tendon splicing was done on all the flexors, in order that the lengthened tendons would permit of the extension of the fingers. The cavity of the wound was filled with sterilized olive oil around the tendons and nerves. Capillary drainage inserted; wound closed, and hand placed in hyper-extended position; held there by splints.

The operation consumed an hour and five minutes, under chloroform. The patient behaved well and reacted properly.



FIG. 1.—Deformity in Case No. 1. Since I failed to secure a good photograph of the arm previous to operation, and not wishing to be without a picture of the arm in its pre-operative position, I had it again taken after the operation in its original mal-position. This figure shows it quite well.

Upon exposing the muscles and tendons their small size was very obvious. In carrying out their isolation some difficulty was experienced in identifying one tendon from the adjacent one. The knife was constantly in use. Cleavage was impossible in the middle of the forearm, for here all semblance of tendon sheaths was gone. The tendons appeared longer than normal, and seemed to encroach upon the muscle substance. On account of the pressure atrophy the muscles were small, firm and fibrous to a marked degree. The musculo-tendon splicing being completed, the hand and fingers placed in a hyper-extended position, I could not but remark upon the long and insignificant-looking threads, and express a very doubtful prognosis. The return of arterial blood into the parts after removing the rubber bandage did not clear away the paleness of the structures, nor my apprehension of their viability. The olive oil was used to prevent, if possible, an immediate gluing together of all the tendons again, and I feel sure of its benefit in this case, to the end desired. The median and ulnar nerves were here and there nodular, and in some places smaller than normal.

Postoperative.—On the third day he developed a temperature which on the evening of the fourth day reached 104.4° F. The wound was dressed and found all right. On the fifth day the temperature was down, but again rose rapidly. He had tympany and extreme tenderness in right iliac and hypogastric regions; leucocytes 30,000; also some diarrhœa. The area was again dressed and found in perfect order. He was then operated upon for appendicitis (acute obstructive variety), and that member was found inflamed, enlarged and obstructed. After operation, pain abated and temperature was lower for a few days, but on the fourth day following this last operation pyrexia and its concomitant symptoms returned, and continued more or less for two weeks, but not alarming. Widal reaction and diazo-reaction tests were both negative, and no abdominal spots appeared, although there were diarrhœa and tympanites. Liquid diet was substituted, and quinine sulphate, gr. iii, every four hours, and hydrargyrum cum creta, gr. ii, every night, administered. By the twenty-third day the patient was sufficiently recovered to return to his home. During his stay in the hospital his arm was dressed frequently and under passive motion slowly improved. The wound healed by

first intention. The fingers could then be easily straightened passively, and almost to a complete degree voluntarily. He could hold his fingers completely shut passively and had considerable flexor power. See Figs. 2, 3, and 4.

The following is the last report (November 28, 1905): "He can straighten the fingers and can close them slightly only. He can hold the hand shut by using the thumb after he has shut it with the other hand. He uses the hand to write and do his school-work, but we do not think he has got as much use of it as you expected; still we think it is gaining all the time." (Figs. 6 and 7.)

CASE II.—Mary H., aged 12 years, weight 80 pounds. Entered Chicago Hospital, November 5, 1904, with the following history, in part, viz.: Ten weeks previously the left humerus was fractured at the junction of the lower and middle thirds. The fragments were soon placed in apposition, and tight splints were bandaged on the entire arm and forearm, and left in this position for seven weeks.

Examination revealed a scar, one-half inch wide and three inches long, extending from the front around to the posterior surface of the arm, three inches above the left elbow joint (Fig. 7). The elbow joint was stiff and tender, and even moderate flexion was impossible. There was a deformity at the wrist and hand, consisting of marked flexion. The proximal phalanges were fully extended, and the two distal segments of the phalanges completely flexed. The forearm showed great atrophy, this being more marked in the region of the flexor bellies. She was unable of herself to adjust this malposition, and any attempt to correct it by forcible manipulation was not only painful, but entirely devoid of even temporary improvement, giving marked evidence of the matting together of the structures of the forearm. Sensations of heat and cold, muscular sense, and tactile sense were lost, except in the index and little fingers, and here they were imperfect and slow.

The hand was colder than its fellow, in all probability showing vasomotor interference.

Operation.—Having applied an Esmarch bandage, an incision was made along the inner border of the biceps for the purpose of exposing the seat of fracture of the humerus and liberating any



FIG. 2.—Showing the extreme limit of voluntary flexion of wrist after operation in case 1.



FIG. 3.—Showing limit of voluntary extension of fingers after operation in case 1.



FIG. 4.—Showing limit of voluntary flexion of fingers after operation in case 1.



FIG. 5.—Case 1, November 28, 1905. Extension over three years after operation.



FIG. 6—Case 1, November 28, 1905. Flexion over three years after operation.



FIG. 7.—Arm before operation in case 2. 1, Transverse scar three inches long running half way around the arm three inches above elbow joint; 2, Atrophy of flexor bellies; 3, Flexion and fixation of wrist; 4, Extension and fixation of proximal phalanges; 5, Acute flexion and fixation of two distal segments of phalanges.

osseous or connective-tissue adhesions which might have formed around the ulnar or median nerves and at the same time determine what damage, if any, had been done to those latter structures. On exposing these nerves, they were found to be normal in appearance, and the bone well united. A second incision was made along the outer surface of the arm, exposing the musculo-spiral nerve, which was thickened and bulbous. These incisions having been closed, the first incision was continued down the forearm and well into the palm. The flexor tendons, having been identified, were individually treated in the following manner: A longitudinal incision, four or five inches long, was made in each muscle and its tendon, cutting across one-half of the tendon from its continuity below, and then transversely severing the muscular portion of the other half above. These corresponding musculo-tendon halves were then temporarily united with fine catgut to maintain their identity. A similar procedure was followed on all the other flexors of the wrist and hand. They were then securely sutured in the extended position by means of three interrupted sutures in each musculo-tendinous structure. Although the wrist and hand were thus liberated, full extension was not possible until the nerves were separated from their surrounding adhesions. The median was atrophied to less than half its normal diameter, below the elbow, and the ulnar nerve showed bulbous enlargements, with atrophied intervals. The wrist and hand were now easily straightened. The skin was closed, and the forearm was drained in several places with strands of silkworm gut. Sterile olive oil was poured over the tendons. The dressings consisted of aseptic gauze next to the skin over which was placed an abundance of iodoform gauze. A plaster-of-Paris cast was then applied and the hand and fingers placed in hyperextension.

The operation was completed in one hour and fifty minutes. Anæsthetics, chloroform and ether. Pulse before operation, 70; during, 108; and after, 88. Hypodermoclysis of normal salt solution at conclusion of operation (500 c.c.).

Results.—Union of skin by first intention. Chromoform catgut No. 0, not absorbed in three weeks.

Report Six Weeks Later.—1. Tenderness in elbow is fast disappearing and flexion and extension are almost complete.

2. More vital activity is gradually coming into the hand.
3. Can immediately tell which finger is touched, showing a return of sensation.
4. Can pick up a small article, like a pin, after trying for a while, showing a return of muscular sense and coördination.
5. Fingers can be straightened and flexed to a considerable extent, and this power is increasing.

It is well to point out that the fracture was of the humerus, and the tight bandaging was on the forearm; the fracture united perfectly, and no damage was done to any of the soft structures surrounding it, but where the bandages were too tightly applied, atrophy, contraction and paralysis of the soft structures ensued.

In regard to fixed state in which the nerves were found, one can readily imagine how easily they could be ruptured by forcible extension of the fingers under an anæsthetic, and irreparable damage done.

The last report was in November, 1904, a year after the operation. Then she had all the motions of the elbow, forearm, wrist, and fingers almost complete, but not as strong as formerly. Sensation had returned to normal apparently, and she can readily distinguish between heat and cold. She no longer burns her fingers. If another case of this nature presents itself for treatment, I should rather choose to resect the bones than splice the tendons. Although the forearm would be shortened, the hand, etc., would be stronger. The tendons and nerves should always be liberated, and I think oil used.

Literature.—Volkmann was the first surgeon to write on this subject and to point out the dangers to muscle following tight bandaging, too-firmly applied splints and Esmarch's bandage too long in use. The damage done by these means became known as "Volkmann's paralysis," "ischemic paralysis," "ischemic atrophy," etc. Inasmuch as the muscles with their tendons and the nerves are the chief structures involved, the title of my paper "Ischemic Muscular Atrophy, Contractures and Paralysis," may not be inappropriate. The older physiologists conducted some experiments on the influence on muscles when the arteries were tied,—i.e.,

giving rise to paralysis. Hallen (1766), Stetson and Lesser. Kuhne concluded (a) loss of irritability, (b) paralysis of the muscle, and (c) rigor of the muscle, to be due to inhibited nutrition.

Carl Ludwig proved that rigor mortis, death of muscle, giving rise to paralysis. Haller (1766), Stetson and Lesser. was due to lack of oxygen.

Kraske has shown that muscles cannot stand the complete absence of the arterial blood current for six hours (Bernays).

The histologic changes have been studied by Keidelberg, Kraske, Lesser, Bernays, Batton, and others. Metchnikoff and Soudakewitsch established the fact that muscular atrophy in the strict sense of the word should be ranged under the group of phenomena caused by phagocytes (collateral).

Fortunately cases of ischemic atrophy and paralysis are rare. In 1888, at the German Society of Surgeons, held at Berlin, it is reported that Lesser saw seven cases; Bardenheuer, four; Helferich, three; and Konig, seven cases.

Dr. A. C. Bernays, of St. Louis, Missouri, published an able and excellent article in the *Boston Medical and Surgical Journal*, May 21, 1900, "On Ischemic Paralysis and Contracture of Muscles," from which I have extracted some of the data in this production. He does not report any of his cases, but makes mention of them. The first seven references here given were compiled by him.

REFERENCES.

- ¹ Lausanne, 1766, p. 544.
- ² W. Kuhne. "On the Movements and Changes of the Contractile Substances," in the *Archives for Anatomy, Physiology, etc.*, edited by Reichert and Du Bois-Reymaud.
- ³ Pitha-Billroth. *System of Surgery*, vii.
- ⁴ Volkmann's *Contributions to Surgery* (*Centralblatt f. Chir.*, 1881, No. 51, etc.).
- ⁵ Kraske. *Central. f. Chir.*, 1879, No. 12.
- ⁶ Lesser. *The Archives for Experimental Pathology and Pharmacy*, vol. viii.
- ⁷ See *Transactions of German Society of Surgeons*, 1888, xi, Berlin.
- ⁸ Jackson. *Boston M. and S. Journal*, July, 1838.

- * Dwight. Boston M. and S. Journal, October, 1838.
- ¹⁰ Gross. Die Krankhaften Geschwülste, Band I, S., 482.
- ¹¹ Page. Lancet, Jan., 1900: "Volkmann's Ischemic Paralysis."
- ¹² Bernays. Boston M. and S. Journal, May 24, 1900.
- ¹³ Wallis. The Practitioner, 1901, p. 429.
- ¹⁴ Metchnikoff and Soudakewitsch: Annales de l'Institut, Paris, Jan., 1892.
- ¹⁵ Donald Fraser. Glasgow Medical Journal, July, 1892, quotes a case of atrophy of the muscles of both arms in a man who had suffered from an accident in which both these members had been stretched for twenty minutes. (Mentioned here for collateral study.)
- ¹⁶ Lemoine. Lyons Medical, Lyons, Dec., 1891, reports the case of a man in whom atrophy of the deltoid biceps, triceps and supinator longus muscles was caused by the compression of a strap used for carrying parcels. (For collateral study.)
- ¹⁷ Littlewood. Letter to Lancet, Jan. 5, 1901, expresses the opinion that the term "ischemic paralysis" is a misnomer, the contraction occurring after elbow injuries in children being really due to the contraction of cicatricial tissue, the result of laceration of the flexor muscles. A swelling in the upper portion of the flexor muscles of the forearm has been present in all cases which have come under his notice. Splint pressure and splint sores are in no way related to the production of the deformity. (I Year-Book, Goul, 1902). (For collateral study.)